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Profile

Dean, Faculty of Life Sciences,

Professor & Head (**Chair**),
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Academic Experience: Abroad

Southern Illinois University, School of Medicine, Springfield, Illinois, USA

Davis Heart and Lung Institute,
Ohio State Univ, Columbus, Ohio, USA

University of California, San Francisco, California, USA

Academic Experience: India

1981	Assistant Professor (Lecturer),Vikram Univ, Ujjain,India
1994	Associate Professor (Reader),Vikram Univ, Ujjain,India
2002	Professor, Vikram University, Ujjain, MP
2014 to	Present Professor & Head (Chair), Vikram University, Ujjain, MP India.
2014 to	present Dean, Faculty of Life Sciences, Vikram Univ, Ujjain

Publications: 60 in International Journals

Citations:	1918 (till October 2015)
h-index	**22**
i10-index	**29**

Research Guidance:

M.Phil. completed	35
Ph.D. completed	06
Ph.D. registered	06
Graduate students	03 (at USA)

Editor in Chief:
Journal of Biochemical and Pharmacological Research, (International journal published from San Jose, CA 95131, USA).

Current Research

Modulation of SIRT3 Gene Expression

SIRT3 is a major mitochondrial deacetylase that has been found to regulate mitochondrial function, bioenergetics and modulation of the response to oxidative stress. SIRT3 also deacetylates numerous components of the electron transport chain, suggesting that SIRT3 could directly suppress ROS production. In this regard, SIRT3 loss increases cellular ROS levels, contributing to genomic and mitochondrial DNA instability. SIRT3 by targeting more than half a dozen key metabolic enzymes can orchestrate coordinated shifts in mitochondrial metabolism, with potential implications for cancer, neurodegeneration and cardiac stress. We are looking for the key mechanism by which SIRT3 gene expression is altered in cells during various conditions including inflammation and oxidative stress.

Gene Expression in Alzheimer's & Parkinson's Diseases & Impairment of Memory

My interests are focused on the mechanism by which amyloid beta and alpha synuclein genes expression and their protein aggregation and translocation within cellular compartments cause pathogenesis of Alzheimer's and Parkinson's diseases in human. I am interested in exploring the Molecular and biochemical mechanisms of pathogenesis of Alzheimer's and Parkinson's Diseases through mutation, gene silencing and transcriptional changes. I am also interested in applying this knowledge on the mechanism of function of amyloid beta peptide on memory and their integration into complex behavioral manifestations during normal and pathologic states. I am also interested on how the brain changes in response to external and internal stimuli. In particular, I am intrigued by how memories are formed, stored and elaborated. Memory is a fundamental biological function and a critical component of our identity. As such, it involves our brain, mind and psyche. In order to understand how memory becomes an integral part of pathologies in neurodegenerative diseases such as Alzheimer's, my studies are focused on the synaptic plasticity, role of glutamate excitotoxicity and genes and epigenetic factors by which normal physiological functions of amyloid peptide becomes pathogenic.

Mitochondria, Aging & Neurodegenerative diseases

Our research program also involve extensive research on mitochondrial bioenergetics, target studies on TCA cycle enzymes, mitochondrial electron transport chain complexes, mitochondrial functions and impairments and apoptosis. We are interested in finding the key mechanism by which mitochondrial impairments leads to neurodegeneration and how mitochondrial reprogramming can be utilized in therapeutic applications in treatment of neurodegenerative and other age-related diseases.

Publications (http://publicationslist.org/mdsparihar)

Citations =1918 (till 25 October 2015)
(http://scholar.google.co.in/citations?user=ohC4O-4AAAAJ&hl=en)

2015

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2. Solanki I, Parihar P, Mansuri, M L, **Parihar M S** (2015) Neurodegenerative Diseases: from available treatments to prospective herbal therapy. **Neurochemistry Intentional** (Elseiver Science Ltd) in press) **IF 3.4**

3. Parihar P, Solanki I, Mansuri, M L, **Parihar M S** (2015) Mitochondrial Sirtuins: Emerging Roles in Metabolic Regulations, Energy Homeostasis and Diseases. **Experimental Gerontology** (Elseiver Science Ltd) 61, 130–141. **IF 3.7**

4. Parihar P, **Parihar M S** (2015) Increase in Hypothalamic Oxidative stress and Mitochondrial Impairment in Streptozotocin treated Diabetic mice. **Cell. Mol. Biol** (in Press)

2014

5. Mansuri, M L, Parihar P, Solanki I, **Parihar M S** (2014) Flavonoids in Modulation of Cell Survival Signalling Pathways. **Genes & Nutrition**, 9(3):400. doi: 10.1007/s12263-014-0400-z. Epub 2014 Mar 30. PMID: 24682883). **IF 3.4**

6. Priyanka Parihar, Dipali Jat, Pedram Ghafourifar, **Parihar M S** (2014) Efficiency of mitochondrially targeted gallic acid in reducing brain mitochondrial oxidative damage. **Cell. Mol. Biol** 60 (2): 35-41. **IF 1.5**

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7. Jat D, Parihar P, Kothari S. C. and **Parihar M S** (2013) Curcumin reduces oxidative damage by increasing reduced glutathione and preventing membrane permeability transition in isolated brain mitochondria. **Cell. Mol. Biol**., 59: OL1899-OL1905. **IF 1.5**

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9. **Parihar M S (2012)** Alpha-synuclein overexpression and inactivation of respiratory chain complex I increase sensitivity of mitochondrial oxidative energy metabolism and neuronal loss: Implications in Parkinson's disease **. J. Comput. Sci. Syst. Biol**. 5:1. **IF 5.63**

2011

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2010

11. **Parihar, M. S**. & Brewer, G. J. (**2010**) Amyloid beta as a modulator of synaptic plasticity. **J Alzheimer's Disease** 22, 741-763. (PMID: 20847424). Cited by 50. **IF 4.2**

12. Parihar A, **Parihar M S**, Nazarewicz, R, Ghafourifar P (**2010**). Importance of cytochrome c redox state in ceramide induced apoptosis in human adenocarcinoma cells. **Biochim. Biophys. Acta** 1800, 646-654. (PMID: 20382204). **IF 4.6**

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15. **Parihar, M.**; Parihar, A.; Fujita, M.; Hashimoto, M.; Ghafourifar, P. (2009) Altered mitochondrial functions by alpha-synuclein overexpression and aggregation in human neuroblastoma cells. **Parkinsonism and Related Disorders** vol. 15 December, 2009. p. S150. DOI: 10.1016/S1353-8020(09)70574-7. ISSN: 1353-8020. **IF 3.9**

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16. **Parihar, M S,** Parihar, A, Fujita W J, Hashimoto M and Ghafourifar, P. (**2008**). Mitochondrial association of alpha-synuclein causes oxidative stress. **Cell Mol. Life Sci**. 65(7-8):1272-84. Cited by 151. **IF 7.04**

17. **Parihar M S,** Elizabeth K and Brewer G J **(2008)** Age-related decreases in NAD(P)H and glutathione cause redox decline before ATP loss during glutamate treatment of hippocampal neurons. **J Neurosci. Res** 86 (10): 2339-52. **IF 3.0**

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19. **Parihar, M S**, Parihar A, Villamena F A, Vaccaro P S, Ghafourifar P (**2008**). Inactivation of mitochondrial respiratory chain complex I leads mitochondrial nitric oxide synthase to become pro-oxidative. **Biochem.Biophys.Res.Commun**. 367, 761-767. **IF 2.6**

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32. Brewer, G. J. **Parihar**, **M. S.,** Jones T. T. (2006) Age-related declines in mitochondrial function during glutamate excitotoxicity. **Alzheimer's & Dementia: J Alzheimer's Assoc**. 2, S479. **IF 10.04**

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35. Parihar, M. S. & Hemnani, T. (2004) Experimental excitotoxicity provokes oxidative damage in mice brain and attenuation by extract of Asparagus racemosus. **J. Neural Transm**. 111, 1-12. PMID: 14714211. **IF 3.05**

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neurotoxicity: implication in neurodegenerative disease. **Current Science** 73, 290-293,1997. Cited by 6. **IF 0.93**

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1995

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INTERNATIONAL BOOKS (Contributed chapters)

59. Hemnani T and **Parihar, M. S.** (1999). An attempt to bypass the bioenergetic defects and oxidative stress in tert-butyl hydroperoxide induced neurotoxicity by pretreatment with a combination of compounds: Implications in neurodegenerative disorders. **Free radicals and Antioxidants** (eds. P. P. Singh, A. K. Pendse, B. S. Bomb, M. K. Barjatiya, Reeta Ghosh; Chaudhary offset Pvt. Ltd. Udaipur) pp. 199-205, 1999.

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